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Maladaptive Perfectionism and the Impact of Fibromyalgia: A Serial Mediation Model Through Sleep Reactivity, Insomnia Severity, and Pain Catastrophizing

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A B S T R A C T

This study aimed to examine the association between maladaptive perfectionism and fibromyalgia impact and to test the serial mediating roles of sleep reactivity, insomnia severity, and pain catastrophizing. This cross-sectional, descriptive-correlational study included 300 adults with physician-diagnosed fibromyalgia residing in Canada. Participants were recruited from rheumatology and chronic pain clinics, community organizations, support groups, and online patient networks. Data were collected using the Discrepancy subscale of the Almost Perfect Scale–Revised, the Ford Insomnia Response to Stress Test, the Insomnia Severity Index, the Pain Catastrophizing Scale, and the Revised Fibromyalgia Impact Questionnaire. Pearson correlations and serial mediation analysis were conducted using IBM SPSS Statistics 29 and PROCESS Model 6. Age, gender, fibromyalgia duration, average pain intensity, and sleep-medication use were controlled. Indirect effects were tested with 10,000 bias-corrected bootstrap resamples. Maladaptive perfectionism significantly predicted sleep reactivity, insomnia severity, pain catastrophizing, and fibromyalgia impact. Sleep reactivity significantly predicted insomnia severity and pain catastrophizing, whereas insomnia severity significantly predicted pain catastrophizing and fibromyalgia impact. Pain catastrophizing was the strongest unique predictor of fibromyalgia impact. The complete serial indirect pathway from maladaptive perfectionism through sleep reactivity, insomnia severity, and pain catastrophizing to fibromyalgia impact was significant. All specific indirect effects were also significant, and the total indirect effect accounted for a substantial proportion of the overall association. The final model explained 69.6% of the variance in fibromyalgia impact. The remaining direct effect of maladaptive perfectionism was significant, indicating partial mediation. Maladaptive perfectionism may increase fibromyalgia burden through heightened stress-related sleep vulnerability, more severe insomnia, and stronger catastrophic interpretations of pain, supporting integrated interventions targeting perfectionistic self-evaluation, sleep disturbance, and pain-related cognition.

Keywords: *Maladaptive perfectionism; fibromyalgia impact; sleep reactivity; insomnia severity; pain catastrophizing; serial mediation.*

1. Introduction

Fibromyalgia is a chronic, multidimensional pain condition characterized by widespread musculoskeletal pain, fatigue, nonrestorative sleep, cognitive difficulties, emotional distress, and substantial interference with physical, occupational, and social functioning. Its clinical burden cannot be explained solely by peripheral tissue damage or a single biomedical abnormality; rather, fibromyalgia is increasingly understood through an integrated biopsychosocial framework in which altered pain processing, sleep disruption, autonomic dysregulation, cognitive-affective vulnerability, behavioral responses, and environmental stress interact over time. Contemporary accounts of chronic pain emphasize that pain intensity and disability are shaped not only by nociceptive input but also by descending modulation, arousal, attention, emotional regulation, and beliefs about the meaning and controllability of symptoms (Trouvin et al., 2022). Accordingly, patients with apparently similar levels of pain may differ markedly in their functional impairment, fatigue, perceived symptom severity, healthcare utilization, and capacity to maintain daily activities. The biobehavioral pain-hygiene perspective similarly proposes that sleep, physical activity, stress exposure, coping behavior, cognitive appraisal, and self-management practices operate as interdependent determinants of chronic pain outcomes rather than as isolated clinical targets (Saravanan & Reagan, 2021). This multidimensional understanding is particularly relevant to fibromyalgia, in which symptom exacerbations frequently occur in the context of stress, disturbed sleep, heightened physiological arousal, and negative interpretations of pain. Evidence-based self-management recommendations therefore increasingly include sleep regulation, graded activity, symptom monitoring, cognitive coping, and digital or remotely delivered support in addition to pharmacological care (Foustoukos et al., 2024). Mind–body interventions have also been proposed as clinically relevant components of treatment for functional and pain-related conditions, including fibromyalgia, because they may influence stress reactivity, emotional regulation, autonomic activation, and symptom appraisal (Islam et al., 2022). Likewise, mindfulness-oriented approaches may reduce the impact of fibromyalgia by changing patients' relationships with painful sensations, distressing thoughts, and functional limitations rather than focusing exclusively on symptom elimination (Gordon et al., 2022).

Within this biopsychosocial framework, personality-related vulnerability may help explain why some individuals experience greater symptom burden than others. Fibromyalgia has been associated with psychological characteristics that can affect autonomic regulation, stress responsivity, coping, and adaptation to persistent symptoms. Personality constructs should not be interpreted as causes of fibromyalgia or as evidence that symptoms are psychologically fabricated; instead, they may function as vulnerability or maintenance factors that shape how individuals respond to pain, fatigue, uncertainty, functional loss, and treatment demands. A narrative review examining personality as a possible biomarker in fibromyalgia proposed that personality-related tendencies may be relevant to autonomic rehabilitation and individualized symptom management (Kulshreshtha, 2023). Similar efforts to clarify the intersection of psychological processes and persistent physical symptoms have emerged in research on post-concussion symptoms and functional neurological disorder, where symptom persistence may involve complex interactions among biological disruption, attention, expectations, arousal, and behavioral adaptation (Mollica et al., 2025). Research on post-COVID neurological complaints has likewise illustrated the need to distinguish genuine symptom burden from overly simplistic biomedical or psychological explanations while considering the role of somatic amplification and symptom-focused distress (Kachaner et al., 2022). Studies of migraine have further shown that behavioral and psychological factors can be clinically relevant even among individuals without formal psychiatric comorbidity (Pistoia et al., 2022). These findings support the broader proposition that psychological vulnerabilities can contribute to the severity and functional consequences of chronic symptoms without invalidating their physiological reality.

Maladaptive perfectionism represents one such vulnerability. Perfectionism is a multidimensional construct involving high personal standards, concerns over mistakes, perceived discrepancies between expectations and performance, excessive self-criticism, fear of negative evaluation, and conditional self-worth. The distinction between adaptive striving and maladaptive evaluative concern is essential because high standards alone do not necessarily produce distress. Maladaptive perfectionism arises when individuals interpret anything short of ideal performance as failure, remain preoccupied with errors, experience persistent dissatisfaction despite accomplishment, and evaluate themselves through rigid

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standards that are difficult or impossible to satisfy. Research among medical and dental students has demonstrated that perfectionism is closely intertwined with anxiety and avoidance-related outcomes, indicating that rigid performance expectations may become harmful when combined with threat sensitivity and self-critical evaluation (Rezaei et al., 2024). Recent work on sleep in medical students has similarly distinguished potentially constructive and harmful dimensions of perfectionism, suggesting that their effects on sleep may depend partly on depression and anxiety (Du, 2026). Although much perfectionism research has focused on academic or occupational functioning, its implications extend to health-related adjustment. Patients with chronic illness may judge themselves harshly for reduced productivity, inability to maintain former roles, inconsistent symptom control, dependence on others, or failure to comply perfectly with treatment plans. In youth with juvenile idiopathic arthritis, perfectionism has been examined as a contributor to internalizing symptoms, illustrating how rigid standards and self-evaluative concerns may intensify distress in the context of chronic pain and functional limitation (Brandelli et al., 2024). A relationship between perfectionism and pain has also been identified among patients with temporomandibular disorders, providing more direct evidence that perfectionistic characteristics may be associated with the experience and interpretation of persistent pain (Xiong et al., 2023).

The relevance of maladaptive perfectionism to fibromyalgia may be especially pronounced because the illness is unpredictable, fluctuating, and difficult to control. Individuals may awaken with severe fatigue despite careful planning, experience pain after seemingly modest activity, or be unable to meet personal, family, and occupational responsibilities at a previously acceptable level. For a person whose self-worth is tied to flawless performance and complete control, these limitations can generate repeated discrepancy experiences: the perceived gap between how one believes one should function and how one is currently able to function. Such discrepancy may increase rumination, self-blame, anxiety, compensatory overexertion, and difficulty disengaging from unmet goals. Broader transdiagnostic research has shown that perfectionistic and compulsive traits may appear across different clinical presentations rather than being restricted to a single diagnosis. For example, obsessive-compulsive symptoms and traits have been investigated among patients with burning mouth syndrome, a chronic pain condition that can involve persistent distress in the absence of straightforward structural explanations (Pellegrini et al., 2025). Perfectionistic cognitive styles are also prominent in disorders involving rigid self-evaluation and overcontrol, as illustrated by narrative evidence concerning eating-disorder symptoms and their evidence-based treatments (Ortiz et al., 2026). Clinical neuroscience perspectives increasingly recognize the value of examining such cognitive-affective characteristics alongside neurophysiological and behavioral processes (Ünal-Aydın et al., 2022). Applied to fibromyalgia, this perspective suggests that maladaptive perfectionism may not influence symptom impact directly alone; it may initiate a sequence of stress-related, sleep-related, and pain-related processes that gradually amplify disability.

Sleep reactivity may constitute the first mechanism in this sequence. Sleep reactivity refers to the extent to which an individual's sleep system becomes disrupted in response to stress, emotional activation, environmental change, or demanding life events. People with high sleep reactivity may develop difficulty falling asleep, repeated awakenings, lighter sleep, or early morning awakening after stressors that produce little sleep disruption in less reactive individuals. Sleep reactivity is therefore considered a vulnerability factor rather than merely an indicator of current insomnia. Maladaptive perfectionism may increase sleep reactivity through anticipatory worry, repetitive review of mistakes, inability to disengage from unfinished tasks, heightened concern about next-day performance, and attempts to exert excessive control over sleep itself. A systematic review of multidimensional perfectionism and sleep disturbance concluded that maladaptive perfectionistic dimensions are more consistently associated with impaired sleep than are high personal standards alone (Stricker et al., 2022). Research among Chinese international students similarly demonstrated an association between negative perfectionism and poorer sleep quality, with the relationship operating through broader psychological processes under stressful conditions (Chen et al., 2022). More generally, a systematic review of personality and insomnia indicates that personality traits can shape vulnerability to insomnia through arousal, emotional instability, coping patterns, and cognitive responses to sleep difficulty (Akram et al., 2023). Psychological predictors of insomnia identified in university populations further suggest that modifiable cognitive-emotional characteristics may serve as prevention targets before sleep disturbance becomes chronic (Ohlsson et al., 2022).

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The transition from sleep reactivity to insomnia severity is consistent with contemporary cognitive models of insomnia. A reactive sleep system may initially produce intermittent sleep disruption during periods of pain, stress, or emotional overload. However, recurrent episodes can become persistent when individuals begin monitoring sleep, worrying about its consequences, extending time in bed, altering daily activity, catastrophizing about next-day impairment, or attempting to force sleep through rigid routines. A systematic review of cognitive factors in insomnia models identified worry, rumination, attentional bias, dysfunctional beliefs, sleep-related monitoring, and perceived loss of control as central processes in the development and maintenance of insomnia (Tang et al., 2023). Network analysis has also linked insomnia-related psychological difficulties to hyperarousal, reinforcing the view that insomnia reflects an interconnected system of cognitive, emotional, and physiological activation rather than insufficient sleep opportunity alone (Zhao et al., 2024). Transdiagnostic research among patients with chronic insomnia has further emphasized vulnerability factors that cross conventional diagnostic boundaries, including emotional dysregulation, repetitive negative thinking, and maladaptive beliefs (Habibi et al., 2023). These mechanisms are likely to be intensified in fibromyalgia because bedtime does not necessarily provide relief from symptoms; pain may interfere with sleep onset, awaken the individual during the night, and increase apprehension about another unrefreshing night. Gender is also clinically relevant because fibromyalgia is diagnosed more frequently in women, while insomnia vulnerability and expression vary across hormonal, reproductive, psychosocial, and aging-related stages. A lifespan review of insomnia has emphasized meaningful gender-related differences in insomnia risk, presentation, and associated burden (Baldi et al., 2025). Consequently, insomnia severity may represent a major pathway through which perfectionism-related arousal becomes translated into greater fibromyalgia impact.

The relationship between sleep and pain is reciprocal and self-reinforcing. Pain can disrupt sleep by increasing physiological arousal, limiting comfortable sleeping positions, and producing nocturnal awakenings, whereas insufficient or fragmented sleep may heighten pain sensitivity, weaken endogenous pain inhibition, impair emotional regulation, and reduce coping resources. Clinical reviews of chronic pain populations indicate that sleep disturbance is not simply a secondary consequence of pain but can independently predict greater pain intensity and poorer functioning (Selvanathan et al., 2021). In fibromyalgia, this reciprocity may be especially consequential because nonrestorative sleep and fatigue are central components of the syndrome. Insomnia can lower the threshold for emotional distress, increase attentional capture by bodily sensations, and make pain appear more threatening and less manageable. Individuals who are exhausted may also have fewer cognitive resources available for reappraisal, pacing, problem solving, and engagement in valued activities. Self-related attitudes may influence this process: structural modeling research has linked self-compassion with sleep quality, emotional distress, and mental well-being, suggesting that a kinder and less punitive stance toward oneself may protect sleep and psychological functioning (Rakhimov et al., 2022). By implication, the harsh self-criticism characteristic of maladaptive perfectionism may have the opposite effect, increasing arousal and diminishing recovery when symptoms interfere with daily goals.

Pain catastrophizing may represent the next mechanism linking insomnia severity to fibromyalgia impact. Pain catastrophizing is characterized by repetitive focus on pain, magnification of its threat, and helplessness regarding one's capacity to manage it. Catastrophizing does not imply that pain is imaginary or exaggerated deliberately; rather, it describes a cognitive-affective response that can intensify attention to pain, increase emotional distress, promote avoidance, and undermine confidence in coping. In fibromyalgia, pain catastrophizing has been shown to mediate relationships involving functional capacity, pain intensity, and depressive symptoms, highlighting its role in translating physical limitations into greater subjective suffering and impairment (Montoro & Galvez-Sánchez, 2022). Insomnia may strengthen catastrophizing by increasing irritability, reducing executive control, impairing cognitive flexibility, and heightening threat appraisal. Evidence from people with opioid use disorder and chronic pain indicates that pain catastrophizing can alter the association between chronic pain and insomnia severity (Baime et al., 2023). Related research in treatment-seeking adults with opioid use disorder has also examined the interrelations among sleep, pain catastrophizing, and pain intensity, supporting the clinical relevance of considering these processes together (Ault et al., 2024). Although opioid-use-disorder populations differ from patients with fibromyalgia, the findings illustrate a broader mechanism in which poor sleep and catastrophic pain cognition may become mutually reinforcing across chronic pain contexts.

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A serial model linking maladaptive perfectionism, sleep reactivity, insomnia severity, pain catastrophizing, and fibromyalgia impact offers several conceptual advantages. First, it distinguishes vulnerability to sleep disruption from the current severity of insomnia. A person may possess a highly reactive sleep system before meeting the threshold for clinically meaningful insomnia, and this vulnerability may become activated by pain, interpersonal stress, perceived failure, or overexertion. Second, the model recognizes that insomnia may influence fibromyalgia impact both directly and indirectly through pain-related cognition. Severe insomnia can worsen fatigue, concentration, mood, and physical functioning, but it may also increase the tendency to interpret pain as uncontrollable and disastrous. Third, the model situates maladaptive perfectionism upstream rather than treating it only as a concurrent correlate of distress. This placement is theoretically plausible because rigid standards, discrepancy experiences, and self-critical rumination may generate the arousal that destabilizes sleep, while chronic sleep disturbance may subsequently impair cognitive-emotional regulation. Fourth, the model integrates personality, sleep, cognition, and functional health within a single explanatory framework. Such integration is compatible with evidence that chronic pain outcomes are shaped by interactions among central modulation, behavioral adaptation, symptom beliefs, and self-management processes rather than by a single linear cause.

Despite growing evidence connecting perfectionism with sleep disturbance, sleep with chronic pain, and pain catastrophizing with fibromyalgia disability, these relationships have generally been examined separately. Existing perfectionism studies have often focused on students, internalizing symptoms, or other chronic pain conditions, while fibromyalgia research has more frequently examined depression, anxiety, mindfulness, or pain catastrophizing without locating these variables within a sequential sleep-vulnerability pathway. Moreover, the distinction between sleep reactivity and insomnia severity has received limited attention in models of fibromyalgia impact. Establishing whether these mechanisms form a statistically coherent sequence may clarify why maladaptive perfectionism is associated with greater illness burden and may identify multiple intervention points. Perfectionistic self-criticism may be addressed through cognitive and compassion-focused strategies; sleep reactivity may be targeted through stress regulation and early sleep intervention; insomnia severity may be treated through cognitive behavioral therapy for insomnia; and pain catastrophizing may be reduced through cognitive restructuring, acceptance-based methods, graded activity, and pain neuroscience education. A model containing these mechanisms may therefore support more individualized care than approaches that treat pain intensity as the only determinant of functional impairment.

Accordingly, the aim of the present study was to examine the association between maladaptive perfectionism and fibromyalgia impact among adults with fibromyalgia in Canada and to test the serial mediating roles of sleep reactivity, insomnia severity, and pain catastrophizing.

2. Methods and Materials

2.1. Study Design and Participants

This study employed a cross-sectional, descriptive-correlational design to examine the relationship between maladaptive perfectionism and the impact of fibromyalgia and to test the serial mediating roles of sleep reactivity, insomnia severity, and pain catastrophizing. The target population consisted of adults with fibromyalgia residing in Canada. Participants were recruited from outpatient rheumatology and chronic pain clinics, fibromyalgia support organizations, community health centers, and online patient communities across several Canadian provinces. Recruitment announcements briefly described the study objectives, eligibility criteria, voluntary nature of participation, and electronic survey procedure. A consecutive and convenience sampling strategy was used because individuals with fibromyalgia are distributed across clinical and community settings and may experience mobility, fatigue, and pain-related barriers that restrict participation in conventional face-to-face research.

A total of 368 individuals initially accessed the online survey. Of these, 29 did not complete the informed consent procedure, 18 did not meet the eligibility criteria, and 21 submitted substantially incomplete questionnaires or response patterns that indicated insufficient engagement. After data screening, the final analytical sample consisted of exactly 300 adults with fibromyalgia. The sample size was considered adequate for the proposed serial mediation model, which included one predictor, three sequential mediators, one outcome variable, and relevant demographic and clinical covariates. The sample also exceeded commonly recommended minimum requirements for regression-based mediation analysis and provided sufficient statistical power to detect small-to-moderate indirect effects using bias-corrected bootstrap confidence intervals.

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Participants were eligible for inclusion when they were at least 18 years of age, currently resided in Canada, were able to read and understand English, had received a diagnosis of fibromyalgia from a physician or qualified healthcare professional, and had experienced fibromyalgia-related symptoms for at least six months. Participants were also required to have access to an internet-connected device and to provide electronic informed consent before entering the survey. Exclusion criteria included a self-reported diagnosis of a severe neurological disorder, active psychotic disorder, bipolar disorder in an acute manic phase, severe cognitive impairment, untreated sleep apnea, or another medical condition that could independently produce widespread pain and severe sleep disturbance. Individuals were also excluded when they were undergoing an acute medical crisis or had undergone major surgery during the preceding three months. These criteria were established to reduce the influence of conditions that could substantially confound the relationships among perfectionism, sleep processes, pain-related cognition, and fibromyalgia impact.

Data were collected through a secure web-based survey platform. Before beginning the questionnaires, participants viewed an information page explaining the purpose of the study, expected duration of participation, potential discomfort associated with answering questions about pain and sleep, confidentiality protections, and their right to discontinue participation without penalty. Participants who agreed to participate provided electronic informed consent and then completed a demographic and clinical information form followed by the study measures. To minimize order effects, the principal questionnaires were presented in a randomized sequence. The survey required approximately 25 to 35 minutes to complete. No personally identifying information was included in the analytical dataset, and electronic data were stored on encrypted, password-protected servers accessible only to the research team. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and received approval from the responsible institutional research ethics board in Canada.

The demographic and clinical information form collected data on age, sex, gender identity, marital status, educational level, employment status, province of residence, duration of fibromyalgia, time since diagnosis, current pain-related medications, use of sleep medication, comorbid physical and psychological conditions, and participation in psychological or physical rehabilitation programs. Participants also rated their average pain intensity during the previous week on an 11-point numerical rating scale ranging from 0, indicating no pain, to 10, indicating the worst imaginable pain. These variables were collected to describe the sample comprehensively and to identify potential covariates that might influence fibromyalgia impact. Age, sex, duration of fibromyalgia, average pain intensity, and current use of sleep medication were considered as potential covariates in the statistical analyses because of their theoretical and clinical associations with sleep disruption and fibromyalgia-related functional impairment.

2.2. Measures

Maladaptive perfectionism was assessed using the Discrepancy subscale of the Almost Perfect Scale–Revised. This instrument evaluates perfectionistic tendencies through dimensions related to high personal standards, preference for order, and perceived discrepancy between personal standards and actual performance. The Discrepancy subscale was selected as the primary indicator of maladaptive perfectionism because it reflects persistent perceptions of failure, dissatisfaction with performance, excessive self-criticism, and the belief that personal achievements do not meet one's own expectations. The subscale contains 12 items rated on a seven-point Likert scale ranging from 1, strongly disagree, to 7, strongly agree. Item scores are summed to produce a total discrepancy score, with higher scores representing greater maladaptive perfectionism. The instrument has demonstrated satisfactory construct validity, internal consistency, and associations with psychological distress, negative affect, self-criticism, and impaired adjustment. In the present study, internal consistency was evaluated using both Cronbach's alpha and McDonald's omega.

Sleep reactivity was measured using the Ford Insomnia Response to Stress Test. This nine-item self-report instrument assesses the degree to which an individual's sleep is disrupted in response to stressful experiences and environmental challenges. The items address situations such as interpersonal conflict, work-related stress, receiving bad news, changes in sleep schedules, and stressful events occurring during the day. Participants indicate the likelihood that each situation would interfere with their sleep using a four-point response scale ranging from 1, not likely, to 4, very likely. Item scores are summed, and higher total scores indicate greater vulnerability to stress-related sleep disturbance. Sleep reactivity is conceptualized as a relatively stable predisposition that increases the probability of

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developing or maintaining insomnia when an individual is exposed to stress. The measure has shown acceptable reliability and predictive validity in clinical and community samples and is particularly relevant to the present model because fibromyalgia-related pain and functional limitations may act as chronic stressors that activate sleep vulnerability.

Insomnia severity was assessed using the Insomnia Severity Index. The Insomnia Severity Index is a seven-item measure designed to evaluate the nature, severity, and consequences of insomnia symptoms during the preceding two weeks. The items assess difficulty initiating sleep, difficulty maintaining sleep, early morning awakening, satisfaction with the current sleep pattern, interference of sleep problems with daily functioning, noticeability of sleep-related impairment to others, and distress or concern caused by sleep difficulties. Each item is rated on a five-point scale ranging from 0 to 4, producing a total score between 0 and 28. Higher scores indicate more severe insomnia symptoms. Total scores are commonly interpreted as indicating no clinically significant insomnia, subthreshold insomnia, moderate clinical insomnia, or severe clinical insomnia. The Insomnia Severity Index has demonstrated strong internal consistency, convergent validity, sensitivity to treatment-related change, and applicability among individuals with chronic pain conditions. In the current study, the continuous total score was used in the mediation analysis rather than diagnostic categories.

Pain catastrophizing was evaluated using the Pain Catastrophizing Scale. This 13-item self-report questionnaire measures exaggerated negative cognitive and emotional responses that may occur during actual or anticipated painful experiences. The measure includes three related dimensions: rumination, which refers to persistent attention to pain-related thoughts; magnification, which reflects an exaggerated perception of the threat associated with pain; and helplessness, which indicates perceived inability to control or manage pain. Participants rate the extent to which they experience each thought or feeling when they are in pain on a five-point scale ranging from 0, not at all, to 4, all the time. The items are summed to create a total score ranging from 0 to 52, with higher scores indicating greater pain catastrophizing. The total score was used as the mediator in the hypothesized serial model because the study focused on the overall cognitive-affective tendency to interpret pain as threatening, uncontrollable, and overwhelming. The scale has shown high internal consistency and significant relationships with pain intensity, emotional distress, disability, healthcare use, and poor treatment outcomes in chronic pain populations.

Fibromyalgia impact was assessed using the Revised Fibromyalgia Impact Questionnaire. This 21-item instrument was developed to evaluate the multidimensional burden of fibromyalgia during the previous seven days. It contains three domains covering physical functioning, overall impact, and symptom severity. The physical functioning domain evaluates difficulties in performing everyday activities, including household tasks, walking, climbing stairs, shopping, and personal care. The overall impact domain assesses the extent to which fibromyalgia interferes with goal attainment and the degree to which participants feel overwhelmed by the condition. The symptom domain evaluates pain, fatigue, stiffness, sleep quality, depression, anxiety, memory difficulties, tenderness, balance problems, and environmental sensitivity. Items are rated using 11-point numerical scales ranging from 0 to 10. Following the standard scoring procedure, weighted domain scores are combined to produce a total score ranging from 0 to 100. Higher scores indicate greater symptom burden, functional impairment, and overall impact of fibromyalgia. The total score was used as the principal dependent variable. The Revised Fibromyalgia Impact Questionnaire has demonstrated strong reliability, construct validity, and sensitivity to clinical differences and treatment effects among individuals with fibromyalgia.

To reduce the potential effects of careless responding, the survey included two instructed-response attention checks distributed across the questionnaires. Cases were flagged for additional review when participants failed both attention checks, completed the survey in an implausibly short period, selected the same response option for nearly all items, or left more than 10% of the principal questionnaire items unanswered. Participants with substantial missing data or clearly invalid response patterns were excluded before the final analysis. For participants with only a small number of missing item responses, scale scores were calculated according to the scoring guidelines of the respective instrument, provided that at least 80% of the items on the relevant scale had been completed.

2.3. Data Analysis

Data analysis was performed using IBM SPSS Statistics, version 29, and the PROCESS macro for SPSS. Before testing the hypotheses, the dataset was examined for accuracy, missing values, univariate and multivariate

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outliers, normality, linearity, homoscedasticity, and multicollinearity. Frequencies, percentages, means, standard deviations, observed ranges, skewness, and kurtosis were calculated to describe the demographic, clinical, and study variables. Internal consistency was assessed using Cronbach's alpha and McDonald's omega coefficients, with values of .70 or higher considered acceptable. The pattern and proportion of missing data were examined before analysis. When item-level missingness was minimal and compatible with the scoring instructions of the instruments, participant-level mean substitution within the relevant scale was used. Cases with substantial missingness across the primary study variables were removed from the analytical dataset.

Univariate outliers were examined through standardized scores, with absolute values greater than 3.29 considered potentially extreme. Multivariate outliers were assessed using Mahalanobis distance at a conservative probability level of $p < .001$. The normality of variable distributions was evaluated through skewness and kurtosis indices, histograms, normal probability plots, and examination of residuals. Linearity and homoscedasticity were assessed using scatterplots of standardized predicted values and standardized residuals. Multicollinearity was evaluated through tolerance statistics and variance inflation factors. Tolerance values below .10 and variance inflation factor values above 10 were regarded as indicators of serious multicollinearity. Because indirect effects often have non-normal sampling distributions, bootstrap estimation was used as the principal method for testing mediation, regardless of whether the observed variables met conventional normality assumptions.

Pearson product-moment correlation coefficients were calculated to examine bivariate relationships among maladaptive perfectionism, sleep reactivity, insomnia severity, pain catastrophizing, and fibromyalgia impact. Independent-samples t tests, one-way analyses of variance, or correlation analyses were used, as appropriate, to explore whether demographic and clinical characteristics were significantly related to the dependent variable. Variables that demonstrated theoretically meaningful and statistically significant associations with fibromyalgia impact were entered as covariates in the mediation model. Age, sex, duration of fibromyalgia, average pain intensity, and use of sleep medication were retained as covariates because they could affect sleep disturbance, pain-related cognition, and functional impairment. Continuous covariates were mean-centered before their inclusion in the model, and categorical variables were dummy coded.

The hypothesized serial mediation model was tested using Model 6 of the PROCESS macro. Maladaptive perfectionism was entered as the independent variable, sleep reactivity as the first mediator, insomnia severity as the second mediator, pain catastrophizing as the third mediator, and fibromyalgia impact as the dependent variable. The model examined whether maladaptive perfectionism was associated with greater fibromyalgia impact directly and indirectly through a theoretically ordered sequence in which maladaptive perfectionism increased vulnerability to stress-related sleep disturbance, greater sleep reactivity contributed to more severe insomnia, insomnia severity intensified catastrophic interpretations of pain, and pain catastrophizing increased the overall functional and symptomatic impact of fibromyalgia.

Ordinary least-squares regression was used to estimate all component paths of the mediation model. The analysis generated estimates for the total effect of maladaptive perfectionism on fibromyalgia impact, the direct effect remaining after inclusion of the mediators, and the specific indirect effects operating through each mediator and combination of mediators. The primary specific indirect effect represented the complete serial pathway from maladaptive perfectionism through sleep reactivity, insomnia severity, and pain catastrophizing to fibromyalgia impact. Additional indirect pathways included maladaptive perfectionism through sleep reactivity alone, insomnia severity alone, pain catastrophizing alone, sleep reactivity followed by insomnia severity, sleep reactivity followed by pain catastrophizing, and insomnia severity followed by pain catastrophizing. Although these additional pathways were estimated, the complete three-mediator serial pathway was considered the principal test of the proposed conceptual model.

Indirect effects were evaluated using 10,000 bootstrap resamples and 95% bias-corrected confidence intervals. An indirect effect was considered statistically significant when its bootstrap confidence interval did not include zero. Unstandardized regression coefficients were reported for the PROCESS estimates, together with standard errors, t values, p values, and confidence intervals. Standardized coefficients were also calculated to facilitate interpretation of the relative magnitude of associations among the study variables. The proportion of variance explained in each endogenous variable was reported using the coefficient of determination. Statistical significance for direct regression paths and

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supplementary analyses was established at a two-tailed alpha level of .05. Because the principal mediation hypotheses were evaluated using bootstrap confidence intervals, conclusions regarding indirect effects were based primarily on the confidence-interval criterion rather than normal-theory significance tests.

To evaluate the robustness of the findings, sensitivity analyses were conducted by estimating the serial mediation model both with and without demographic and clinical covariates. A further analysis was performed after excluding participants who reported regular use of prescribed sleep medication, because pharmacological treatment could influence the relationship between sleep reactivity and insomnia severity. The results of the primary model were considered robust when the direction and statistical significance of the complete serial indirect effect remained consistent across these alternative specifications. Because the study used a cross-sectional design, the ordering of the mediators was based on the theoretical model rather than observed temporal precedence. Accordingly, the mediation findings were interpreted as evidence of statistically consistent indirect associations and not as definitive proof of causal or longitudinal mechanisms.

3. Findings and Results

The final analytical sample consisted of 300 adults with a physician-confirmed diagnosis of fibromyalgia residing in Canada. Participants ranged in age from 20 to 72 years, with a mean age of 48.17 years (SD = 10.72). A total of 258 participants identified as women (86.0%), 37 identified as men (12.3%), and five identified as gender-diverse or nonbinary (1.7%). With respect to geographical distribution, 118 participants resided in Ontario (39.3%), 51 in Quebec (17.0%), 43 in British Columbia (14.3%), 36 in Alberta (12.0%), 14 in Manitoba (4.7%), 12 in Saskatchewan (4.0%), 10 in Nova Scotia (3.3%), seven in New Brunswick (2.3%), five in Newfoundland and Labrador (1.7%), two in Prince Edward Island (0.7%), and two in Yukon or the Northwest Territories (0.7%). Regarding marital status, 179 participants were married or living in a common-law relationship (59.7%), 54 were single and had never married (18.0%), 52 were divorced or separated (17.3%), and 15 were widowed (5.0%). Fifty-five participants had completed secondary education or less (18.3%), 110 had completed college, technical, or vocational education (36.7%), 93 held a bachelor's degree (31.0%), and 42 held a graduate or professional degree (14.0%). Seventy-four participants were employed full-time (24.7%), 52 were employed part-time (17.3%), 95 were receiving disability benefits or were on medical leave (31.7%), 32 were unemployed (10.7%), and 47 were retired (15.7%). The mean duration of fibromyalgia symptoms was 8.41 years (SD = 6.77; range = 0.50–32.00 years), whereas the mean time elapsed since formal diagnosis was 6.17 years (SD = 5.49; range = 0.25–27.00 years). Participants reported a mean pain intensity of 6.68 out of 10 during the preceding week (SD = 1.52), indicating a generally moderate-to-severe level of ongoing pain. A total of 219 participants reported current use of analgesic or pain-management medication (73.0%), 168 reported use of antidepressant medication (56.0%), and 109 reported regular use of prescribed or nonprescribed sleep medication (36.3%). In addition, 188 participants reported a current or previous diagnosis of an anxiety or depressive disorder (62.7%), 122 were participating in a physical rehabilitation or structured exercise program (40.7%), and 81 were receiving psychological or counselling services at the time of data collection (27.0%). These categories were not mutually exclusive because participants could report more than one medication, comorbid condition, or treatment modality.

Before the principal analyses were conducted, the data were examined for completeness, distributional properties, influential observations, linearity, homoscedasticity, independence of residuals, and multicollinearity. Item-level missingness among the 300 retained cases was low, accounting for 0.62% of all expected item responses, and no participant had more than 10% missing responses on any principal measure. Scale scores for participants with limited item-level missingness were therefore calculated using person-mean substitution within the relevant scale, provided that at least 80% of the items had been completed. Standardized scores did not exceed the absolute value of 3.29 for any principal variable, and examination of Mahalanobis distance at $p < .001$ did not identify any remaining multivariate outlier requiring exclusion. Skewness values ranged from −0.42 to 0.31, and kurtosis values ranged from −0.67 to 0.29, indicating no substantial departure from univariate normality. Inspection of histograms, normal probability plots, scatterplots, and standardized residuals supported the assumptions of approximate normality, linearity, and homoscedasticity. Tolerance statistics ranged from .46 to .83, and variance inflation factors ranged from 1.21 to 2.19, indicating that multicollinearity was not problematic. The Durbin–Watson statistic for the final fibromyalgia-impact regression

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equation was 1.96, supporting the independence of residuals. Accordingly, all 300 retained cases were included in the descriptive, correlational, regression, and bootstrap mediation analyses.

Table 1

Descriptive Statistics, Internal Consistency Coefficients, and Correlations Among the Principal Study Variables

Variable	Possible range	Observed range	Mean	SD	Cronbach's α	McDonald's ω	1	2	3	4	5
1. Maladaptive perfectionism	12–84	19–82	54.72	11.46	.93	.93	—				
2. Sleep reactivity	9–36	10–36	24.88	5.76	.88	.89	.451***	—			
3. Insomnia severity	0–28	3–28	17.93	5.82	.91	.91	.403***	.588***	—		
4. Pain catastrophizing	0–52	2–51	29.14	10.47	.94	.94	.391***	.445***	.596***	—	
5. Fibromyalgia impact	0–100	20.40–94.80	62.36	15.79	.92	.93	.478***	.522***	.6		

As shown in Table 1, participants reported relatively elevated levels of all principal study variables. The mean maladaptive-perfectionism score was 54.72 (SD = 11.46) on a possible range of 12 to 84, indicating that perceived discrepancy between personal standards and actual performance was prominent within the sample. The mean sleep-reactivity score was 24.88 (SD = 5.76), suggesting that many participants experienced a substantial vulnerability to sleep disruption when exposed to stress. The mean Insomnia Severity Index score was 17.93 (SD = 5.82), which fell within the conventional range associated with moderate clinical insomnia. The mean pain-catastrophizing score was 29.14 (SD = 10.47), indicating considerable rumination, magnification, and helplessness in relation to pain. The mean Revised Fibromyalgia Impact Questionnaire score was 62.36 (SD = 15.79), reflecting a substantial overall burden of fibromyalgia symptoms, functional limitations, and interference with daily activities. All measures demonstrated strong internal consistency, with Cronbach's alpha coefficients ranging from .88 to .94 and McDonald's omega coefficients ranging from .89 to .94. The zero-order correlations were positive and statistically significant in every case. Maladaptive perfectionism was positively associated with sleep reactivity ($r = .451$, $p < .001$), insomnia severity ($r = .403$, $p < .001$), pain catastrophizing ($r = .391$, $p < .001$), and fibromyalgia impact ($r = .478$, $p < .001$). Sleep reactivity was positively correlated with insomnia severity ($r = .588$, $p < .001$), pain catastrophizing ($r = .445$, $p < .001$), and fibromyalgia impact ($r = .522$, $p < .001$). Insomnia severity was strongly associated with pain catastrophizing ($r = .596$, $p < .001$) and fibromyalgia impact ($r = .638$, $p < .001$). The strongest bivariate relationship was observed between pain catastrophizing and fibromyalgia impact ($r = .699$, $p < .001$), indicating that participants who interpreted pain in a more threatening, uncontrollable, and overwhelming manner tended to experience markedly greater symptom burden and functional impairment. The magnitude and direction of these correlations were consistent with the proposed serial mediation model and supported proceeding to multivariable path analysis.

Table 2

Regression Coefficients for the Serial Mediation Model Predicting Sleep Reactivity, Insomnia Severity, Pain Catastrophizing, and Fibromyalgia Impact

Dependent variable	Predictor	B	SE	β	t	p	95% CI for B
Sleep reactivity	Maladaptive perfectionism	0.226	0.026	.450	8.69	< .001	[0.175, 0.277]
Insomnia severity	Maladaptive perfectionism	0.079	0.024	.156	3.29	.001	[0.032, 0.126]
Insomnia severity	Sleep reactivity	0.469	0.048	.464	9.77	< .001	[0.375, 0.563]
Pain catastrophizing	Maladaptive perfectionism	0.110	0.038	.120	2.89	.004	[0.035, 0.185]
Pain catastrophizing	Sleep reactivity	0.173	0.078	.095	2.22	.027	[0.020, 0.326]
Pain catastrophizing	Insomnia severity	0.804	0.091	.447	8.84	< .001	[0.625, 0.983]
Fibromyalgia impact	Maladaptive perfectionism	0.173	0.061	.126	2.84	.005	[0.053, 0.293]
Fibromyalgia impact	Sleep reactivity	0.279	0.127	.102	2.20	.029	[0.029, 0.529]
Fibromyalgia impact	Insomnia severity	0.608	0.151	.224	4.03	< .001	[0.311, 0.905]
Fibromyalgia impact	Pain catastrophizing	0.715	0.079	.474	9.05	< .001	[0.560, 0.870]

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Table 2 presents the individual regression paths constituting the hypothesized serial mediation model. After adjustment for age, gender, fibromyalgia duration, average pain intensity, and sleep-medication use, maladaptive perfectionism significantly and positively predicted sleep reactivity (B = 0.226, SE = 0.026, β = .450, t = 8.69, p < .001, 95% CI [0.175, 0.277]). This coefficient indicated that each one-point increase in maladaptive perfectionism was associated with an estimated 0.226-point increase in sleep reactivity, controlling for the demographic and clinical covariates. Maladaptive perfectionism also retained a significant direct association with insomnia severity when sleep reactivity and the covariates were included in the equation (B = 0.079, SE = 0.024, β = .156, t = 3.29, p = .001, 95% CI [0.032, 0.126]). Sleep reactivity was a stronger predictor of insomnia severity (B = 0.469, SE = 0.048, β = .464, t = 9.77, p < .001, 95% CI [0.375, 0.563]), demonstrating that individuals whose sleep was more vulnerable to stress reported substantially more severe difficulty initiating sleep, maintaining sleep, and functioning during the day. In the equation predicting pain catastrophizing, maladaptive perfectionism remained significant (B = 0.110, SE = 0.038, β = .120, p = .004), as did sleep reactivity (B = 0.173, SE = 0.078, β = .095, p = .027). Insomnia severity was the strongest predictor of pain catastrophizing in this equation (B = 0.804, SE = 0.091, β = .447, t = 8.84, p < .001, 95% CI [0.625, 0.983]), suggesting that more severe insomnia was associated with a pronounced increase in repetitive, threatening, and helpless appraisals of pain. In the final outcome equation, pain catastrophizing demonstrated the largest unique association with fibromyalgia impact (B = 0.715, SE = 0.079, β = .474, t = 9.05, p < .001, 95% CI [0.560, 0.870]). Insomnia severity also made an independent contribution to fibromyalgia impact (B = 0.608, SE = 0.151, β = .224, t = 4.03, p < .001), while sleep reactivity remained a smaller but significant predictor after the other mediators were controlled (B = 0.279, SE = 0.127, β = .102, t = 2.20, p = .029). Maladaptive perfectionism retained a statistically significant direct effect on fibromyalgia impact after all three mediators and covariates had been entered (B = 0.173, SE = 0.061, β = .126, t = 2.84, p = .005). The persistence of this direct effect indicated partial rather than complete mediation, meaning that maladaptive perfectionism was associated with fibromyalgia impact both through the proposed sleep- and pain-related mechanisms and through additional mechanisms not represented in the model.

Table 3

Bootstrap Estimates of the Specific and Total Indirect Effects of Maladaptive Perfectionism on Fibromyalgia Impact

Indirect pathway	Indirect effect	Boot SE	Boot LLCI	Boot ULCI	Result
Maladaptive perfectionism → Sleep reactivity → Fibromyalgia impact	0.063	0.021	0.026	0.107	Significant
Maladaptive perfectionism → Insomnia severity → Fibromyalgia impact	0.048	0.018	0.015	0.087	Significant
Maladaptive perfectionism → Pain catastrophizing → Fibromyalgia impact	0.079	0.027	0.029	0.135	Significant
Maladaptive perfectionism → Sleep reactivity → Insomnia severity → Fibromyalgia impact	0.064	0.017	0.035	0.101	Significant
Maladaptive perfectionism → Sleep reactivity → Pain catastrophizing → Fibromyalgia impact	0.028	0.012	0.006	0.055	Significant
Maladaptive perfectionism → Insomnia severity → Pain catastrophizing → Fibromyalgia impact	0.045	0.017	0.015	0.082	Significant
Maladaptive perfectionism → Sleep reactivity → Insomnia severity → Pain catastrophizing → Fibromyalgia impact	0.061	0.016	0.033	0.096	Significant
Total indirect effect	0.388	0.055	0.288	0.500	Significant

The bootstrap mediation results presented in Table 3 supported all seven specific indirect pathways represented in the serial mediation model. The indirect relationship between maladaptive perfectionism and fibromyalgia impact through sleep reactivity alone was significant, with an indirect effect of 0.063 and a 95% bootstrap confidence interval that did not include zero (Boot SE = 0.021, 95% CI [0.026, 0.107]). This result indicated that greater maladaptive perfectionism was associated with greater vulnerability to stress-related sleep disturbance, which was subsequently associated with greater fibromyalgia impact. The specific indirect pathway through insomnia severity alone was also significant (indirect effect = 0.048, Boot SE = 0.018, 95% CI [0.015, 0.087]), indicating that maladaptive perfectionism was related to fibromyalgia impact partly because it was associated with more severe insomnia symptoms. The indirect effect operating through pain catastrophizing alone was significant and somewhat larger

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(indirect effect = 0.079, Boot SE = 0.027, 95% CI [0.029, 0.135]), demonstrating that maladaptive perfectionism was associated with more catastrophic cognitive and emotional responses to pain and, consequently, with greater fibromyalgia-related impairment. The two-mediator pathway in which maladaptive perfectionism predicted sleep reactivity, which then predicted insomnia severity and subsequently fibromyalgia impact, was significant (indirect effect = 0.064, Boot SE = 0.017, 95% CI [0.035, 0.101]). The pathway from maladaptive perfectionism through sleep reactivity and pain catastrophizing was also significant (indirect effect = 0.028, Boot SE = 0.012, 95% CI [0.006, 0.055]), although it was the smallest of the specific indirect effects. Similarly, the pathway passing sequentially through insomnia severity and pain catastrophizing was significant (indirect effect = 0.045, Boot SE = 0.017, 95% CI [0.015, 0.082]). Most importantly, the complete hypothesized serial pathway from maladaptive perfectionism to sleep reactivity, from sleep reactivity to insomnia severity, from insomnia severity to pain catastrophizing, and from pain catastrophizing to fibromyalgia impact was statistically significant (indirect effect = 0.061, Boot SE = 0.016, 95% CI [0.033, 0.096]). The confidence interval for this complete serial indirect effect remained entirely above zero, providing support for the proposed ordering of the mediators. The total indirect effect combining all seven pathways was 0.388 (Boot SE = 0.055, 95% CI [0.288, 0.500]). Thus, a substantial proportion of the association between maladaptive perfectionism and fibromyalgia impact was transmitted through sleep reactivity, insomnia severity, and pain catastrophizing, either independently or in sequence.

Table 4

Model Summary Statistics for the Four Regression Equations in the Serial Mediation Analysis

Dependent variable	R	R^2	Adjusted R^2	RMSE	F	df	p
Sleep reactivity	.491	.241	.225	5.08	15.51	6, 293	< .001
Insomnia severity	.679	.461	.448	4.32	35.68	7, 292	< .001
Pain catastrophizing	.706	.498	.484	7.52	36.09	8, 291	< .001
Fibromyalgia impact	.834	.696	.686	8.85	73.77	9, 290	< .001

As reported in Table 4, each regression equation was statistically significant and accounted for a meaningful proportion of variance in its respective dependent variable. The equation predicting sleep reactivity from maladaptive perfectionism and the five demographic and clinical covariates was significant, $F(6, 293) = 15.51$, $p < .001$, and explained 24.1% of the variance in sleep reactivity. After adjustment for the number of predictors, the model retained an adjusted R^2 of .225, indicating that maladaptive perfectionism and the covariates collectively explained approximately one quarter of individual differences in vulnerability to stress-induced sleep disturbance. The insomnia-severity equation was also significant, $F(7, 292) = 35.68$, $p < .001$, accounting for 46.1% of the variance in insomnia severity. This considerable increase in explained variance was consistent with the strong contribution of sleep reactivity to insomnia symptoms. The pain-catastrophizing equation explained 49.8% of the variance, adjusted $R^2 = .484$, $F(8, 291) = 36.09$, $p < .001$, demonstrating that maladaptive perfectionism, sleep reactivity, insomnia severity, and the covariates jointly accounted for nearly half of the variability in catastrophic pain-related cognition. The final fibromyalgia-impact equation demonstrated the greatest explanatory power, $R = .834$, $R^2 = .696$, adjusted $R^2 = .686$, $F(9, 290) = 73.77$, $p < .001$. Thus, maladaptive perfectionism, sleep reactivity, insomnia severity, pain catastrophizing, and the demographic and clinical covariates jointly explained 69.6% of the observed variance in fibromyalgia impact. The total unadjusted effect of maladaptive perfectionism on fibromyalgia impact was $B = 0.561$, $SE = 0.069$, $t = 8.13$, $p < .001$, 95% CI [0.425, 0.697]. After the mediators were introduced, the direct effect decreased to $B = 0.173$, $SE = 0.061$, $t = 2.84$, $p = .005$, 95% CI [0.053, 0.293]. The total indirect effect was 0.388, meaning that approximately 69.2% of the total association between maladaptive perfectionism and fibromyalgia impact was statistically transmitted through the combined mediating pathways, while approximately 30.8% remained as a direct association after the three mediators and covariates were controlled. Sensitivity analyses conducted without covariates and after excluding the 109 participants who regularly used sleep medication produced the same direction and pattern of statistical significance for the complete serial indirect effect, indicating that the principal mediation finding was not dependent on the selected covariate specification or regular sleep-medication use.

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Figure 1

Standardized Path Coefficients for the Serial Mediation Model Linking Maladaptive Perfectionism to Fibromyalgia Impact Through Sleep Reactivity, Insomnia Severity, and Pain Catastrophizing

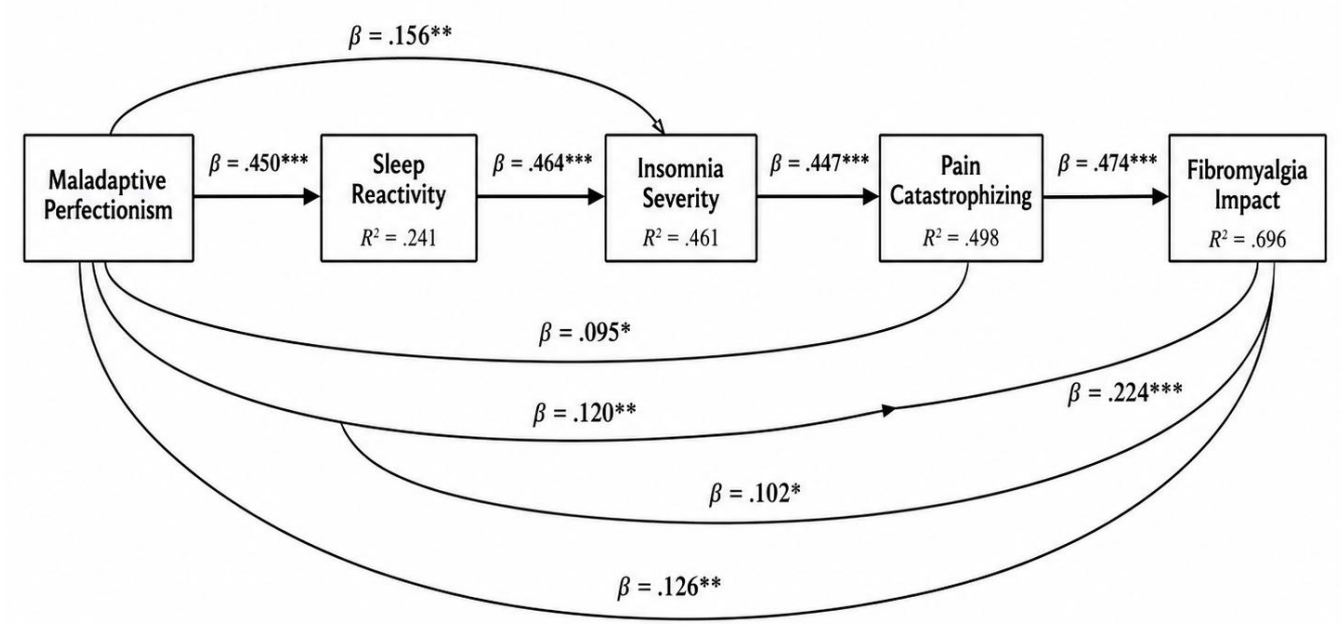

Figure 1 depicts the standardized coefficients for the hypothesized serial mediation model. Maladaptive perfectionism demonstrated a significant positive association with sleep reactivity (β = .450, p < .001), and sleep reactivity was positively associated with insomnia severity (β = .464, p < .001). Insomnia severity, in turn, was significantly associated with pain catastrophizing (β = .447, p < .001), while pain catastrophizing demonstrated the strongest direct association with fibromyalgia impact (β = .474, p < .001). In addition to this complete sequential pathway, maladaptive perfectionism retained significant direct paths to insomnia severity (β = .156, p = .001), pain catastrophizing (β = .120, p = .004), and fibromyalgia impact (β = .126, p = .005). Sleep reactivity also retained significant direct associations with pain catastrophizing (β = .095, p = .027) and fibromyalgia impact (β = .102, p = .029), whereas insomnia severity remained independently associated with fibromyalgia impact (β = .224, p < .001). The pattern of standardized paths indicated that maladaptive perfectionism was associated with greater fibromyalgia impact through a progressive process involving increased susceptibility of sleep to stress, intensification of insomnia symptoms, and escalation of catastrophic pain-related cognition. At the same time, the remaining direct effect of maladaptive perfectionism showed that the sleep-reactivity, insomnia, and pain-catastrophizing sequence did not account for the entire relationship. Overall, the regression and bootstrap findings supported a statistically significant partial serial mediation model in which sleep reactivity, insomnia severity, and pain catastrophizing represented interconnected mechanisms linking maladaptive perfectionism with the functional and symptomatic impact of fibromyalgia.

4. Discussion and Conclusion

The present study examined the relationship between maladaptive perfectionism and fibromyalgia impact and tested whether sleep reactivity, insomnia severity, and pain catastrophizing serially mediated this association among adults with fibromyalgia in Canada. The findings supported the proposed conceptual model. Maladaptive perfectionism was positively associated with sleep reactivity, insomnia severity, pain catastrophizing, and fibromyalgia impact, and all three mediators were significantly related to the overall functional and symptomatic burden of fibromyalgia. The complete serial indirect pathway was statistically significant, indicating that higher maladaptive perfectionism was associated with greater vulnerability of sleep to stress, which was related to more severe insomnia, greater pain catastrophizing, and ultimately greater fibromyalgia impact. The direct effect of maladaptive perfectionism remained

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significant after the mediators and demographic and clinical covariates were controlled, indicating partial rather than complete mediation. The final model explained 69.6% of the variance in fibromyalgia impact, suggesting that the combined contribution of perfectionistic self-evaluation, sleep vulnerability, insomnia symptoms, and catastrophic pain-related cognition was substantial.

The positive association between maladaptive perfectionism and fibromyalgia impact suggests that rigid standards, perceived failure to meet personal expectations, and persistent self-criticism may be clinically relevant to how patients experience and manage fibromyalgia. Fibromyalgia imposes fluctuating limitations that can make it difficult to maintain consistent productivity, family responsibilities, social participation, exercise routines, and self-care behaviors. Individuals characterized by maladaptive perfectionism may interpret these unavoidable inconsistencies as evidence of personal inadequacy rather than as consequences of a chronic and unpredictable condition. This interpretation may intensify emotional distress, promote overexertion on relatively good days, and produce self-blame when symptoms worsen. The finding is compatible with the view that personality-related characteristics may function as biomarkers or vulnerability indicators in fibromyalgia by shaping autonomic responses, coping behaviors, and adjustment to chronic symptoms (Kulshreshtha, 2023). It is also consistent with evidence from temporomandibular disorders showing that perfectionism is meaningfully related to pain experiences in another persistent musculoskeletal pain population (Xiong et al., 2023). Research among young people with juvenile idiopathic arthritis has similarly indicated that perfectionism can contribute to internalizing symptoms in the context of chronic pain and physical limitations (Brandelli et al., 2024). Together, these findings suggest that maladaptive perfectionism may increase illness burden when patients evaluate their functioning against inflexible standards that do not accommodate changes in pain, energy, or physical capacity.

The relationship between maladaptive perfectionism and fibromyalgia impact can also be understood in light of the distinction between high standards and evaluative concerns. High personal standards are not necessarily pathological and may support treatment adherence or purposeful activity when accompanied by flexibility. In contrast, maladaptive perfectionism involves excessive concern about mistakes, dissatisfaction with performance, perceived discrepancy between expectations and achievement, and conditional self-acceptance. Research among medical and dental students has shown that perfectionism is closely associated with anxiety and procrastination, indicating that the pursuit of flawless performance can become immobilizing when accompanied by fear of failure and negative self-appraisal (Rezaei et al., 2024). Evidence concerning perfectionism and sleep in medical students also suggests that maladaptive dimensions may exert harmful effects through depression and anxiety (Du, 2026). Although the populations differ from adults with fibromyalgia, the underlying cognitive process may be comparable: an individual maintains demanding expectations, perceives current performance as inadequate, and remains mentally preoccupied with correcting the discrepancy. In fibromyalgia, the inability to control pain, sleep, fatigue, and daily functioning perfectly may create a continuous source of perceived failure, thereby increasing the overall psychological and functional impact of the condition.

The significant association between maladaptive perfectionism and sleep reactivity supported the first stage of the proposed serial mechanism. Individuals with stronger perfectionistic concerns may be more vulnerable to stress-related sleep disruption because they have difficulty disengaging from unfinished tasks, perceived errors, interpersonal evaluation, and concerns about next-day performance. The present finding is consistent with a systematic review demonstrating that maladaptive perfectionistic dimensions are associated with sleep disturbance more consistently than are high personal standards alone (Stricker et al., 2022). It is also supported by evidence that negative perfectionism is related to poorer sleep quality through psychological mechanisms under stressful circumstances (Chen et al., 2022). Personality-focused insomnia research has emphasized that individual differences in emotional reactivity, self-evaluation, and coping may predispose some people to persistent sleep difficulties (Akram et al., 2023). In the context of fibromyalgia, the combination of perfectionistic cognitive arousal and pain-related bodily arousal may make the sleep system particularly reactive. A patient may go to bed while reviewing incomplete responsibilities, worrying about the effects of poor sleep on the next day, or criticizing herself for being unable to function normally. These responses may prolong physiological activation at precisely the time when cognitive and somatic deactivation are necessary for sleep onset.

Sleep reactivity significantly predicted insomnia severity, providing support for the transition from vulnerability to

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established sleep disturbance. This result is theoretically consistent with cognitive models in which stress-related sleep disruption becomes chronic through worry, monitoring, dysfunctional beliefs, and compensatory behaviors. Cognitive factors such as rumination, attentional bias toward signs of wakefulness, catastrophic expectations regarding sleep loss, and attempts to control sleep have been identified as central maintenance mechanisms of insomnia (Tang et al., 2023). Network evidence also indicates that psychological symptoms related to insomnia are closely linked to hyperarousal, suggesting that insomnia emerges from interacting cognitive, affective, and physiological processes (Zhao et al., 2024). Transdiagnostic vulnerability factors, including repetitive negative thinking and emotional dysregulation, have likewise been implicated in chronic insomnia (Habibi et al., 2023). For adults with fibromyalgia, repeated nights of stress-responsive sleep disturbance may be reinforced by pain-related awakenings and fear of being unable to cope the following day. Over time, the bed and nighttime period may become associated with effort, frustration, vigilance, and anticipated failure rather than rest. The mean insomnia severity score in the present sample was within the moderate clinical range, emphasizing that sleep disturbance was not a peripheral complaint but a major component of the participants' illness burden.

The association between insomnia severity and fibromyalgia impact was also significant after pain catastrophizing and the other predictors were controlled. This finding is consistent with evidence that sleep disturbance contributes independently to chronic pain outcomes and should not be treated solely as a consequence of pain (Selvanathan et al., 2021). Poor sleep may increase pain sensitivity, impair descending pain inhibition, reduce emotional regulation, intensify fatigue, and diminish the cognitive resources required for effective coping. Mechanistic accounts of chronic pain emphasize the contribution of central and descending modulation to variations in pain severity and functional impairment (Trouvin et al., 2022). The present findings also align with the biobehavioral pain-hygiene model, which conceptualizes sleep, stress, activity, cognition, and pain as mutually influential elements of a broader self-regulatory system (Saravanan & Reagan, 2021). In fibromyalgia, even modest sleep disruption may have wide-ranging effects because fatigue, cognitive difficulty, and nonrestorative sleep are already central features of the syndrome. Insomnia may therefore increase fibromyalgia impact through both direct physiological consequences and indirect effects on mood, activity, concentration, and pain appraisal.

Insomnia severity was strongly associated with pain catastrophizing, supporting the next link in the proposed serial model. Sleep deprivation and fragmented sleep may reduce cognitive flexibility, increase negative emotional reactivity, and make painful sensations appear more threatening and less controllable. When individuals are exhausted, they may find it more difficult to redirect attention away from pain, use balanced reappraisal, tolerate uncertainty, or recall previous experiences of successful coping. The present result is consistent with evidence that pain catastrophizing moderates the relationship between chronic pain and insomnia severity among people with opioid use disorder (Baime et al., 2023). Related research has also demonstrated meaningful interrelationships among sleep, pain catastrophizing, and pain intensity in treatment-seeking adults with opioid use disorder (Ault et al., 2024). Although these samples differ diagnostically from patients with fibromyalgia, the convergence of results supports the broader proposition that disturbed sleep and catastrophic pain cognition are closely connected across chronic pain conditions. The present findings extend this evidence by locating insomnia before pain catastrophizing within a theoretically ordered model, suggesting that insomnia may contribute to the cognitive amplification of pain.

Pain catastrophizing was the strongest unique predictor of fibromyalgia impact in the final regression equation. This result indicates that the way patients interpret and respond cognitively to pain may be particularly important for understanding differences in functional impairment and symptom burden. Catastrophizing can increase selective attention to pain, magnify anticipated harm, promote avoidance, reduce confidence in self-management, and strengthen feelings of helplessness. These responses may restrict activity beyond what is physiologically necessary and may contribute to deconditioning, social withdrawal, dependence, and reduced participation in valued roles. The finding closely aligns with evidence that pain catastrophizing mediates relationships among functional capacity, depression, and pain intensity in patients with fibromyalgia (Montoro & Galvez-Sánchez, 2022). It is also compatible with behavioral and psychological research in migraine, which has shown that cognitive-affective characteristics influence pain-related outcomes even in the absence of formal psychiatric comorbidity (Pistoia et al., 2022). The strength of the association observed in the current study suggests that pain catastrophizing may represent a

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proximal mechanism through which more distal vulnerabilities, including perfectionism and sleep disturbance, become translated into greater fibromyalgia-related disability.

The significant complete serial indirect effect provided the most important support for the proposed model. The results suggested that maladaptive perfectionism may increase stress-related sleep vulnerability, which may contribute to clinically meaningful insomnia, intensify catastrophic appraisals of pain, and increase the overall impact of fibromyalgia. This pathway is conceptually coherent because perfectionistic individuals may experience greater cognitive arousal when symptoms interfere with responsibilities, while insufficient sleep may progressively weaken their ability to regulate threatening pain-related thoughts. The finding extends previous research by integrating personality, sleep reactivity, insomnia severity, pain cognition, and functional impact within one model. Existing research has generally examined only selected parts of this sequence, such as perfectionism and sleep (Stricker et al., 2022), insomnia-related cognitive processes (Tang et al., 2023), or pain catastrophizing and fibromyalgia functioning (Montoro & Galvez-Sánchez, 2022). The present model demonstrates how these previously separate lines of research may be connected.

The significant shorter indirect pathways also indicated that the proposed mechanisms were not dependent exclusively on the full serial chain. Maladaptive perfectionism was indirectly related to fibromyalgia impact through sleep reactivity alone, insomnia severity alone, and pain catastrophizing alone. Significant two-mediator pathways were also identified through sleep reactivity and insomnia severity, sleep reactivity and pain catastrophizing, and insomnia severity and pain catastrophizing. These results suggest that several overlapping mechanisms may operate simultaneously. For example, stress-reactive sleep may worsen daytime fatigue and functioning even before severe insomnia develops, whereas maladaptive perfectionism may foster catastrophic pain appraisal directly through intolerance of limitation and perceived loss of control. The persistence of a significant direct effect further indicates that other mechanisms were not captured. These may include anxiety, depressive symptoms, fear of movement, experiential avoidance, autonomic dysregulation, reduced self-compassion, and ineffective activity pacing. The protective relevance of self-compassion is supported by evidence linking a kinder attitude toward oneself with better sleep quality, lower emotional distress, and improved well-being (Rakhimov et al., 2022). Conversely, the self-punitive stance associated with maladaptive perfectionism may contribute to fibromyalgia impact through pathways extending beyond sleep and catastrophizing.

The findings have implications for contemporary approaches to fibromyalgia management. Reviews of self-management strategies emphasize the value of integrated programs addressing sleep, physical activity, symptom interpretation, stress management, and sustained engagement in treatment (Foustoukos et al., 2024). Mindfulness-based symptom-management approaches may help patients observe pain and self-critical thoughts without automatically responding through avoidance or escalation (Gordon et al., 2022). Mind–body interventions may similarly reduce physiological arousal and improve adaptation across fibromyalgia and related functional disorders (Islam et al., 2022). The current results suggest that such interventions may be strengthened when they explicitly address maladaptive perfectionism, particularly rigid expectations regarding productivity, symptom control, treatment adherence, and recovery. Interventions need not discourage meaningful goals; rather, they can help patients replace inflexible standards with realistic, compassionate, and context-sensitive goals that accommodate symptom variability. The gender composition of the present sample also underscores the importance of attending to gender-related patterns in insomnia vulnerability and symptom burden across the lifespan (Baldi et al., 2025).

More broadly, the findings support a non-reductionist interpretation of fibromyalgia. Psychological variables should not be used to dismiss symptoms as unreal or to attribute responsibility for illness to the patient. Research on persistent symptoms in functional neurological, post-concussion, and post-infectious conditions demonstrates the complexity of interactions among biological, cognitive, behavioral, and contextual factors (Kachaner et al., 2022; Mollica et al., 2025). Similarly, psychological traits observed in chronic pain syndromes, including obsessive-compulsive or perfectionistic tendencies, should be understood as potential modifiers of symptom experience rather than as evidence against physiological pathology (Pellegrini et al., 2025). A biopsychosocial model permits recognition of the biological reality of pain while also identifying modifiable psychological and behavioral processes that can reduce disability. The present results therefore support an integrated interpretation in which maladaptive perfectionism, sleep reactivity, insomnia, and

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catastrophizing contribute to fibromyalgia impact without being regarded as its sole causes.

Several limitations should be considered when interpreting the findings. The cross-sectional design did not establish temporal precedence or causality, even though the ordering of the mediators was theoretically justified. It therefore remains possible that greater fibromyalgia impact increases insomnia, pain catastrophizing, or perfectionistic concerns, or that reciprocal relationships operate among the variables. All principal constructs were measured using self-report questionnaires, which may have increased shared-method variance and may have been affected by memory, current mood, social desirability, or response style. The use of convenience and online recruitment may have produced selection bias because individuals who participate in patient communities or internet-based research may differ from patients receiving care in other settings. The sample was predominantly female and English-speaking, limiting generalizability to men, gender-diverse individuals, French-speaking Canadians, and culturally diverse populations. Diagnoses and exclusion criteria were based primarily on participant reports rather than standardized clinical examinations or medical-record verification. Objective sleep measures, inflammatory or autonomic indicators, medication doses, and detailed information regarding psychiatric comorbidity were not included. Finally, although the model accounted for a large proportion of variance, unmeasured factors such as depression, anxiety, pain acceptance, physical activity, socioeconomic stress, trauma history, and treatment access may have influenced the observed relationships.

Future studies should use longitudinal and prospective designs to determine whether maladaptive perfectionism predicts later increases in sleep reactivity, insomnia, catastrophizing, and fibromyalgia impact. Repeated assessments and ecological momentary methods could clarify how daily stress, self-critical thoughts, sleep quality, and pain interact within individuals over time. Experimental and intervention studies are needed to test whether reducing perfectionistic concerns or sleep disturbance produces subsequent reductions in catastrophizing and functional impairment. Future research should combine self-report instruments with actigraphy, polysomnography, daily sleep diaries, quantitative sensory testing, and autonomic or neuroendocrine indicators. Recruitment should include larger proportions of men, gender-diverse adults, French-speaking Canadians, rural residents, Indigenous populations, and individuals from varied cultural and socioeconomic backgrounds. Alternative serial orders and reciprocal models should be compared rather than assuming a single directional sequence. Researchers should also examine potentially protective variables, including self-compassion, psychological flexibility, pain acceptance, social support, adaptive goal adjustment, and effective activity pacing. Latent-variable modeling could reduce measurement error, while person-centered analyses might identify subgroups characterized by distinct combinations of perfectionism, sleep vulnerability, and pain-related cognition.

Assessment and treatment of fibromyalgia should include routine evaluation of maladaptive perfectionism, stress-related sleep vulnerability, insomnia severity, and pain catastrophizing rather than focusing exclusively on pain intensity. Clinicians should explore whether patients hold inflexible expectations regarding productivity, caregiving, exercise, treatment adherence, or symptom control and should help them establish flexible standards that accommodate fluctuations in pain and fatigue. Early identification of sleep reactivity may permit preventive intervention before intermittent sleep disruption develops into persistent insomnia. Patients with clinically meaningful insomnia should be offered evidence-based behavioral sleep treatment integrated with pain management. Cognitive and acceptance-based techniques can be used to reduce rumination, magnification, helplessness, and fear-based avoidance, while compassion-focused strategies may reduce punitive self-evaluation. Interdisciplinary programs should coordinate psychological care, sleep treatment, graded physical activity, medical management, and education regarding the reciprocal relationships among stress, sleep, and pain. The overall clinical objective should be to improve functioning and self-management without implying that fibromyalgia symptoms are imagined or caused solely by personality.

Authors' Contributions

Authors contributed equally to this article.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

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Declaration of Interest

The authors report no conflict of interest.

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Ethics Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

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